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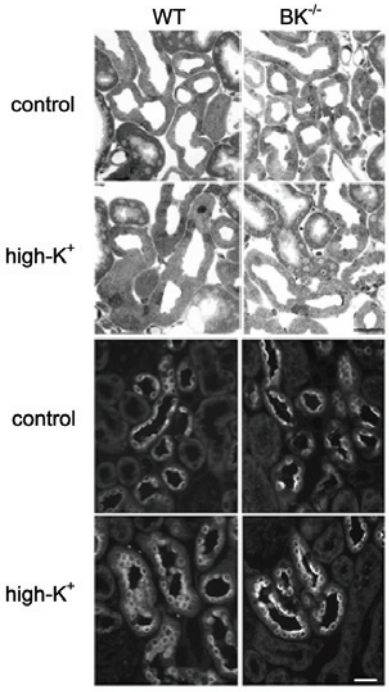
BK channels and ROMK

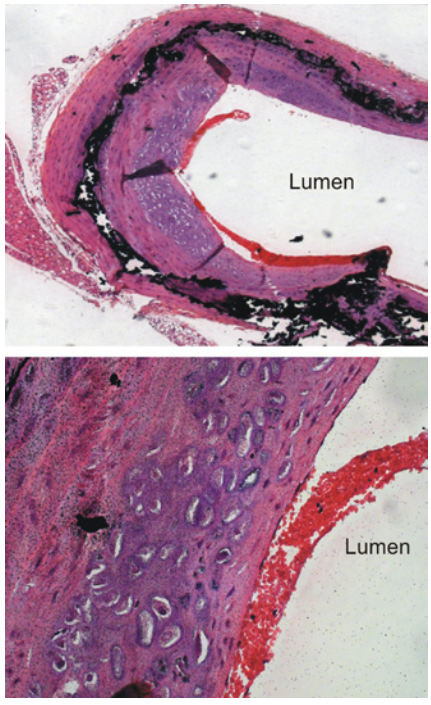
The kidney regulates potassium (K) homeostasis by both absorption and secretion of the filtered ion. Two K channels are known to exist in the distal nephron, the ROMK channel and the BK channel, the latter being calcium activated. Both of these channels are present in the collecting tubule, where K secretion occurs in response to increases in urine flow rate. As they report in this issue, Rieg *et al.* studied the contribution of each of these channels to K secretion. Mice with deletion of the BK channel



had no increase in the rate of K excretion as urine flow rate increased, whereas wild-type mice exhibited the expected effect. Interestingly, changing K in the diet resulted in equivalent K excretion in both wild-type mice and mutant mice lacking the BK channel. However, animals lacking the BK channel had higher aldosterone levels. These animals had greater expression of the ROMK channel. These studies demonstrate that the BK channel is critical for flow-induced K secretion, but that the ROMK channel can compensate for its absence. Thus, K homeostasis is maintained in the mutant mice by increased plasma aldosterone and upregulated ROMK. **See page 566.**

Endochondral bone in arterial calcification

Arterial calcification is emerging as one of the major problems in end-stage renal disease. Studies of its mechanism show the type of calcium mineral formed, and the cause: it is largely due to specific cells located in the media of the arteries. These findings indicated that arterial calcification might resemble bone formation, suggesting that some vascular cells could convert to bone-like cells. Neven *et al.* tested



this hypothesis by inducing a variety of cell markers using the arteries of rats with chronic renal failure and human transplant donors. Remarkably, the rats with chronic kidney failure expressed markers of chondrocytes as well as specific proteins secreted by chondrocytes in calcifying tissues. The more severe the calcification, more similar was the matrix surrounding the calcified tissues to that of typical chondrocytes. Human aorta specimens showing mild to moderate media calcification also showed expression of chondrocyte markers. These studies raise the interesting question of whether the aortic smooth muscle is capable of differentiating into a chondrocyte-like cell, which could cause calcification. **See page 574.**

Iron sucrose versus ferric gluconate

One component of the anemia of chronic renal failure is iron deficiency. Intravenous iron is often used to treat the condition. Recently, many clinicians have replaced iron dextran with iron sucrose or gluconate, because sucrose or gluconate has lower short-term toxicity. However, studies in animals have suggested that these two agents, especially iron sucrose, might be nephrotoxic. In a new paper, Agarwal *et al.* studied 12 patients with advanced chronic kidney disease. The patients were randomized to receive the same dose of either drug 1 week apart. Patients had significantly more proteinuria after receiving iron sucrose than after receiving ferric gluconate. Proteinuria increased within 15 minutes of the infusion. Other markers of proximal tubular toxicity, such as the urine *N*-acetyl-β-D-glucosaminidase/creatinine ratio, were the same after patients received iron sucrose and ferric gluconate. **See page 638.**